

Cardiac Tamponade: Impact of Pathophysiology on Clinical Practice

Roger L. Royster, M.D., Scott R. Coleman, D.O., Karl M. Richardson, M.D.

Cardiac tamponade and constrictive pericarditis are pericardial compressive syndromes of the myocardium. They have different pathophysiology, neuroendocrine response, and clinical signs and symptoms as compared with cardiac syndromes that cause cardiac dilation, like congestive heart failure, despite similar increases in intracardiac pressure.^{1–4} Cardiac tamponade has been studied in animal models for over a half century.^{5,6} However, animal research may not accurately reflect the pathophysiology of tamponade in humans. Although difficult to study in clinical trials, multiple case reports and case series over the years have added to our knowledge and ability to care for these extremely sick patients.^{7,8} This review of cardiac tamponade correlates animal and human research, case reports, and theories into a present-day understanding of the pathophysiology of the disease and assists anesthesiologists with the clinical management of these patients, including controversial areas like positive-pressure ventilation and the effectiveness of chest compressions during cardiopulmonary resuscitation (CPR).

Clinical Signs and Symptoms and Diagnostic Testing

Classic signs of cardiac tamponade are increased jugular venous pressure, distant heart sounds, and hypotension (Beck's triad) resulting from pericardial fluid preventing right-heart diastolic filling; muffled heart sounds; and, ultimately, decreased cardiac output. Additionally, the increase in systemic venous pressure may result in a paradoxical rise in jugular venous filling during inspiration (Kussmaul's sign) due to pericardial compression of the right atrium. Sympathetic activation results in tachycardia, which is often present with hypotension, but hypertension or normotension can be present due to systemic vasoconstriction in patients who are chronically

hypertensive.⁹ Dyspnea occurs due to pulmonary pooling of blood and activation of pulmonary stretch receptors and C fibers that respond to increases in interstitial fluid and pressure.¹⁰

Chest radiography typically demonstrates a globally dilated cardiac silhouette, often without pulmonary edema. Although, in animal studies, interstitial pulmonary edema increases with pulmonary pooling of blood in tamponade, alveolar edema was absent by extravascular thermal volume and histological measurements.^{6,11} In patients having dyspnea, pulmonary edema is also a rare event during tamponade in humans, despite similar increases in diastolic pressures which causes pulmonary edema in congestive heart failure.⁴ One theory is the differences in atrial natriuretic peptide (ANP) and B-type natriuretic peptide (BNP) secretion, which is elevated in cardiac dilation in congestive heart failure and promotes diuresis and increases in pulmonary capillary permeability, while ANP and BNP (without significant chamber stretch) remain normal in cardiac tamponade and therefore maintain normal capillary permeability despite increases in capillary hydrostatic pressure.¹²

An electrocardiogram can demonstrate an alternating QRS axis with each heartbeat due to the heart moving within a fluid-filled pericardium (electrical alternans), and ST segment changes may be present due to pericardial irritation.

An echocardiogram provides a definitive diagnosis of the presence of a pericardial effusion.¹³ As pericardial pressure increases, the right atrium will collapse first, at the end of diastole, as the atrial volume and pressure decrease (fig. 1A).¹⁴ Greater increases in pericardial pressure then cause right ventricular diastolic collapse in early diastole, when the ventricular pressure is lowest, as the chamber begins to fill (fig. 1B). Higher pericardial pressure causes right atrial and then right ventricular collapse throughout

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Abbreviations: ANP, atrial natriuretic peptide; BNP, brain natriuretic peptide; CPR, cardiopulmonary resuscitation; E wave, passive ventricular filling wave



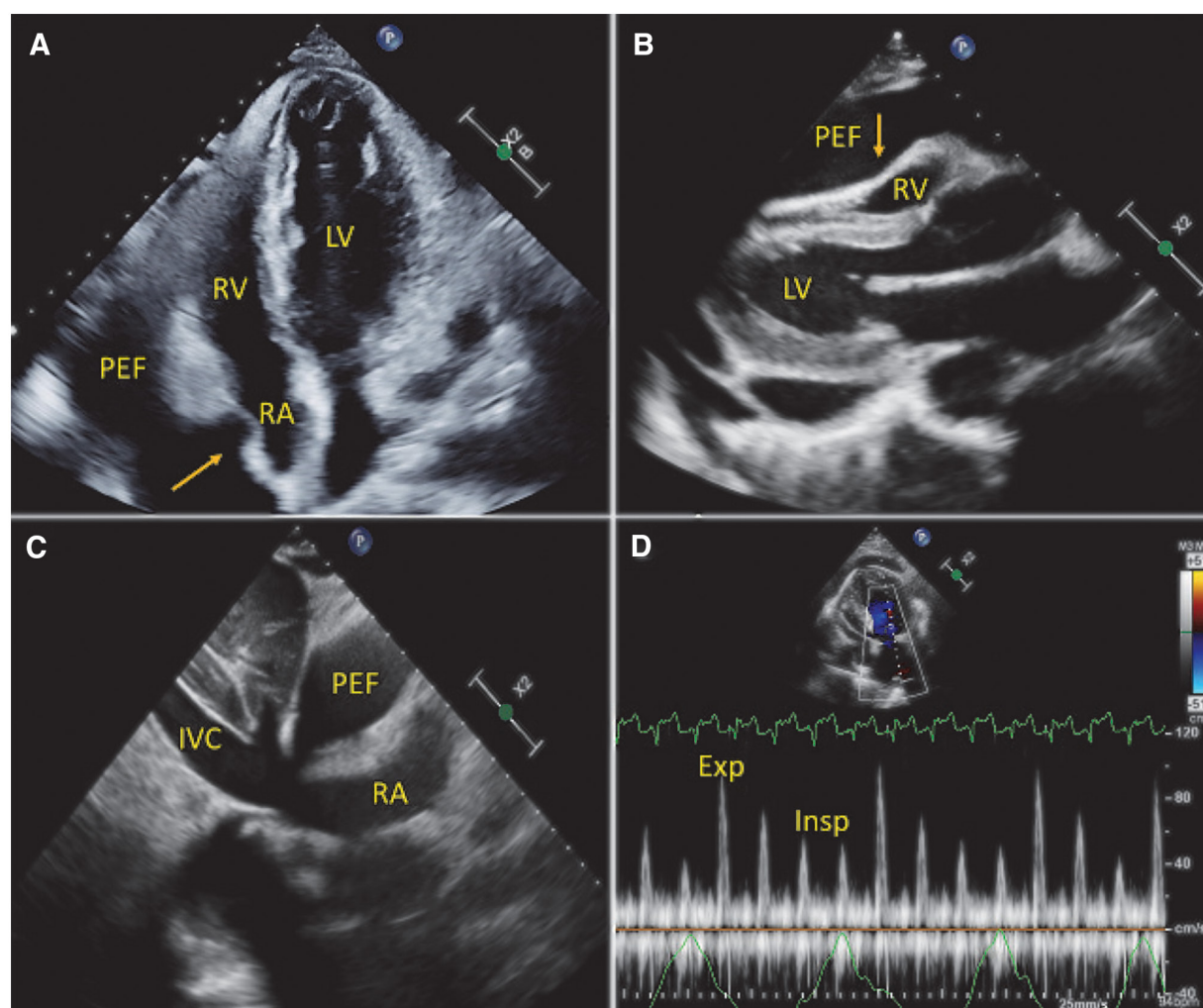


Fig. 1. (A) Transthoracic echocardiogram apical four-chamber view showing right atrial (RA) diastolic collapse (arrow). (B) Parasternal long-axis view showing right ventricular (RV) diastolic collapse (arrow). (C) Subcostal view showing an enlarged inferior vena cava (IVC). (D) Pulse wave Doppler at the mitral valve, illustrating respirophasic inflow variation. During spontaneous inspiration (Insp), increased filling of the RV occurs at the expense of left ventricular (LV) filling, and diastolic flow across the mitral valve (passive ventricular filling wave [E wave]) is decreased. The opposite occurs during expiration as LV filling is enhanced.¹⁴ Exp, expiration; PEF, pericardial effusion.

diastole and, eventually, through much of the cardiac cycle. Usually, the echocardiogram also shows a dilated inferior vena cava with lack of inspiratory diastolic collapse due to high systemic venous pressure (fig. 1C); however, this may not be present in low-pressure tamponade.

Tricuspid valve passive ventricular filling wave (E wave) inflow velocity typically varies with the respiratory cycle, with an inspiratory increase and expiratory decrease of as much as 60% suggestive of significantly increased pericardial pressure affecting right ventricular filling (table 1).^{15,16} With pericardial compression as left ventricular inflow is compromised, the variation in mitral valve E wave inflow velocity rises significantly, with a reduction with inspiration of greater than 25% suggestive of tamponade physiology. Expiration causes a reversal of this reduction in velocity, with

an increase in mitral valve E wave velocity and enhanced left ventricular filling of as much as 22% (fig. 1D).¹⁴ Hepatic vein diastolic flow reversal is also frequently found during cardiac tamponade.¹⁶

Spontaneous Ventilation and Pulsus Paradoxus

During normal spontaneous ventilation, inspiration reduces intrathoracic pressure, increases the pressure gradient for venous return to the right heart, and increases right-heart stroke volume and pulmonary arterial blood flow while at the same time reducing pulmonary venous flow to the left heart, left ventricular stroke volume, and systolic arterial pressure.¹⁷ The opposite physiologic effects happen during expiration with reduced venous return to the right heart, increased venous return to the left heart, and increased

Table 1. Impact of Spontaneous and Positive-pressure Ventilation on Mitral and Tricuspid Valve Peak Flow Velocity and Paradoxical Pulse with and without Cardiac Tamponade

	Mitral Valve	Tricuspid Valve	Paradoxical Pulse
Spontaneous ventilation without tamponade			< 6 mmHg
Inspiration	↓ < 10%	↑ < 25%	
Expiration	↑	↓	
Spontaneous ventilation with tamponade			> 10 mmHg
Inspiration	↓↓ 25–65%	↑↑ 58–85%	
Expiration	↑↑ 22%	↓↓	
Positive-pressure ventilation without tamponade			Reverse paradox
Inspiration	↑	↓	
Expiration	↓	↑	
Positive-pressure ventilation with tamponade			Reduced or absent
Inspiration	↑	↓	
Expiration	↓	↑	

Adapted from Royster *et al.*¹⁵

systolic arterial pressure, which results in a normal respiratory variation in systolic arterial pressure or a normal “paradoxical” pulse. Kussmaul first described this decrease in systolic arterial pressure with respiration more than 100 yr ago as “pulsus paradoxus” by simply palpating the pulse in a patient with constrictive pericarditis.¹⁸ However, a paradoxical pulse is more common in cardiac tamponade.⁸ With spontaneous breathing, a paradoxical pulse may be present and is considered normal with a decrease in systemic arterial pressure of less than 6 mmHg with inspiration. With cardiac tamponade, however, the decrease in systemic arterial pressure from a reduction in left ventricular stroke volume is exaggerated and is defined as greater than 10 mmHg (fig. 2).²

Another concept that supports the pathophysiology of the paradoxical pulse is ventricular interdependence (fig. 3).¹⁴ Rising pericardial pressure causes increasing degrees of constraint on the cardiac chambers and intracardiac volumes. With inspiration and increased right-heart diastolic filling and an accompanying reduction in left-sided venous return, the interventricular septum moves leftward, further reducing left ventricular filling and left ventricular stroke volume. Expiration and the resultant increased left-sided filling allow the septum to move back toward the right, which promotes greater left ventricular filling and further supports pulsus paradoxus. A further reduction in left ventricular contractility, driven by a concomitant reduction in coronary blood flow with tamponade, remains controversial.^{19,20}

Pathophysiology and Compensatory Mechanisms

A classic study of patients with pericardial effusion illustrates the hemodynamic changes in humans that result in three phases as fluid accumulates in the pericardium and causes pericardial pressure to increase (table 2).⁸ The pericardial pressure exerts passive pressure on the cardiac chambers, first affecting the diastolic phase of the right cardiac

chambers. As long as pericardial pressure remains lower than right-heart diastolic pressure, with the transmural pressure (intracardiac minus pericardial pressure) remaining positive, there is no effect on intracardiac volume nor a reduction in cardiac output (phase I). A paradoxical pulse of some degree was present in only 31% of patients. However, as the pericardial pressure rises and begins to equilibrate with right-heart diastolic pressure, and the transmural pressure nears zero, extrinsic compression of the right cardiac chambers occurs, restriction of right-heart filling begins, and cardiac output decreases. A paradoxical pulse was present in 55% of these patients (phase II). When pericardial pressure equilibrates with left ventricular diastolic pressure, as measured by pulmonary capillary wedge pressure, and left ventricular diastolic transmural pressure becomes zero, left ventricular diastolic chamber compression occurs, and cardiac output reduction becomes severe with significant hypotension. The paradoxical pulse was present in 85% of these patients (phase III).

After pericardiocentesis, phase I patients had 270 to 1,860 ml of fluid removed, phase II patients had 200 to 2,400 ml removed, and phase III patients had 250 to 1,750 ml removed from the pericardium. Patients in this study had both chronic and acute pericardial effusion. Chronic effusion usually develops slowly, is larger at presentation, and usually results in a less stiff and more compliant pericardial space, while acute effusion develops rapidly into a more stiff, smaller, and less compliant pericardial space (fig. 4). The rapidity of accumulation can affect the pace at which a patient progresses through these three phases of tamponade.

Compensatory physiologic changes occur to help offset the reduction in systemic venous return and reduction in cardiac output in phases II and III. During cardiac tamponade, spontaneous ventilation during inspiration reduces pericardial pressure while increasing intra-abdominal pressure due to diaphragmatic excursion.

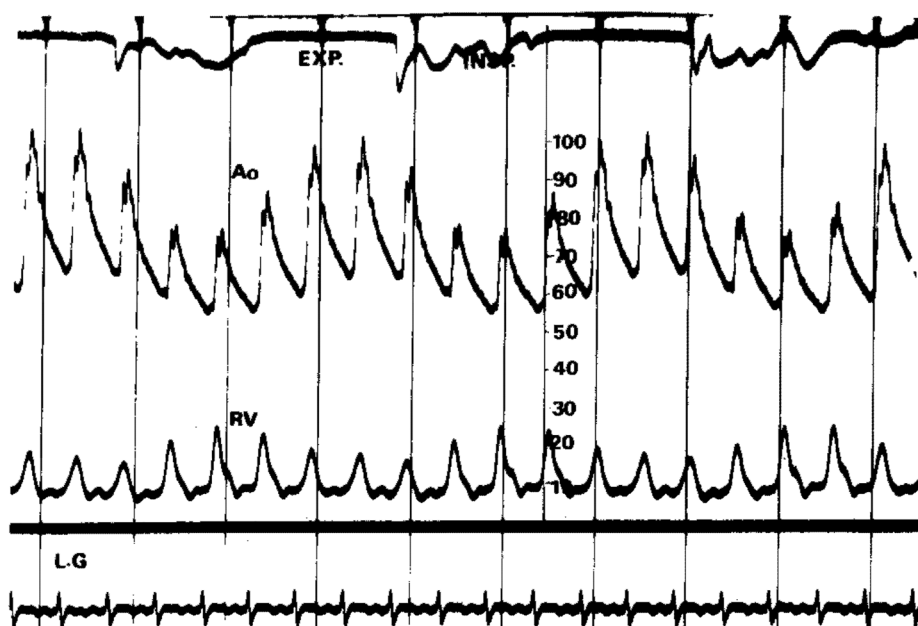


Fig. 2. Aortic (Ao) and right ventricular (RV) pressure during cardiac tamponade in a patient. Ao pressure is increased during expiration (EXP.), while RV pressure is decreased. There is a marked decrease in Ao pressure and an increase in RV pressure during inspiration (Insp), showing a pulsus paradoxus of 25 mmHg.²

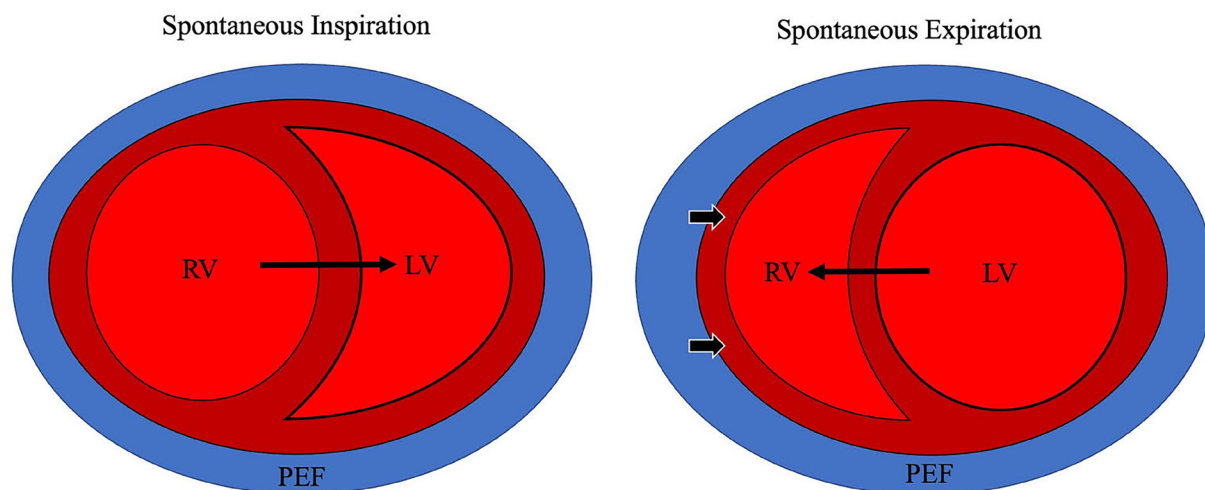


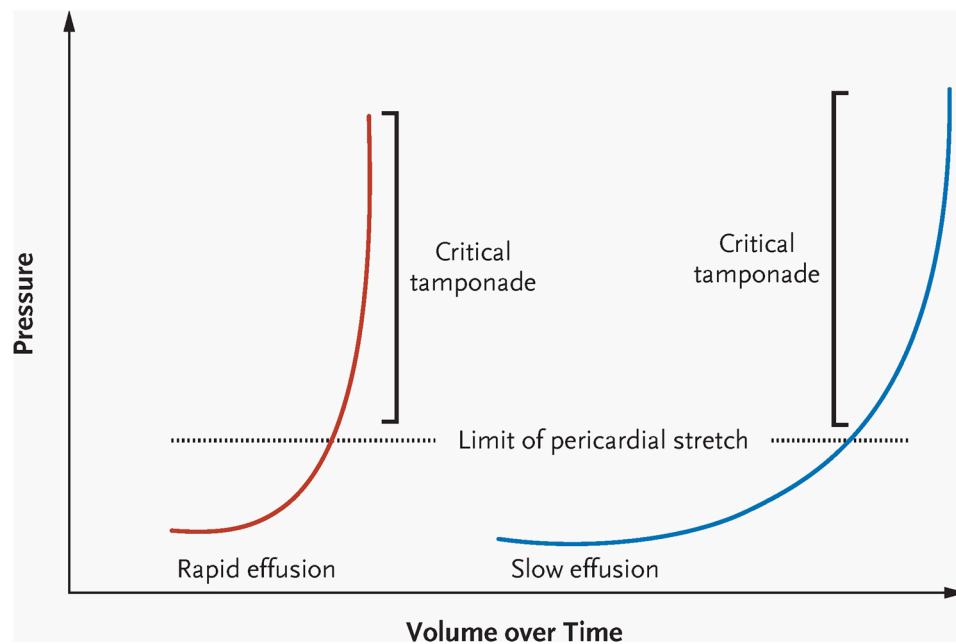
Fig. 3. Ventricular interdependence. During spontaneous inspiration, right ventricular (RV) filling is increased. As the ventricles exist within a fixed pericardial space, RV filling occurs at the expense of the left ventricle (LV), causing an interventricular septal shift toward the left (arrow) and restricting LV stroke volume. During spontaneous expiration, the opposite phenomenon occurs, causing the interventricular septum to shift toward the RV. Leftward arrowheads signify the compressive effects of the pericardial effusion (PEF) on the RV. In cardiac tamponade, differential chamber filling during the respiratory cycle is responsible for a decline in systolic blood pressure greater than 10 mmHg with inspiration (pulsus paradoxus).¹⁴

These pressure changes can help maintain the venous pressure gradient and blood flow to the right heart and pulmonary circulation. Additionally, sympathetic activation increases heart rate, contractility, and systemic

vascular resistance, which also increase systemic and right-heart venous return, increases cardiac output, may reduce heart size and pericardial pressure, and increases mean arterial pressure.

Table 2. Three Phases of Hemodynamics at Baseline and Post Pericardiocentesis in 77 Patients with Pericardial Effusion/Tamponade

		Mean Pericardiac Pressure (mmHg)	Mean Right Atrial Pressure (mmHg)	Mean Pulmonary Artery Wedge Pressure (mmHg)	Cardiac Output (l/min)	Inspiratory Fall in Arterial Systolic Pressure (mmHg)
Phase 1	Baseline	6 ± 0.4	10 ± 5	13 ± 7	5.8 ± 1.8	12 ± 9
Mean pericardiac pressure < mean right atrial pressure < mean pulmonary artery wedge pressure, n = 16	After pericardiocentesis	0 ± 3*	7 ± 6*	12 ± 7	6.0 ± 1.8	9 ± 6
Phase 2	Baseline	9 ± 5	10 ± 5	15 ± 4	5.2 ± 1.5	16 ± 8
Mean pericardiac pressure = mean right atrial pressure < mean pulmonary artery wedge pressure, n = 13	After pericardiocentesis	-2 ± 2*	3 ± 5*	11 ± 8	6.3 ± 2.0*	8 ± 4*
Phase 3	Baseline	16 ± 7	16 ± 7	17 ± 6	4.4 ± 2.0	26 ± 13
Mean pericardiac pressure = mean right atrial pressure = mean pulmonary artery wedge pressure, N = 48	After pericardiocentesis	0 ± 3*	7 ± 5*	9 ± 5*	7.2 ± 2.3*	9 ± 5*

Adapted from Reddy *et al.*⁸**P* < 0.01 comparing baseline *versus* after pericardiocentesis pressure.**Fig. 4.** Pericardial pressure–volume curves are shown, where the volume increases slowly or rapidly over time. (*Left*) rapidly increasing pericardial fluid first reaches the limit of the pericardial reserve volume (the initial flat segment) and then quickly exceeds the limit of pericardial stretch, causing a steep rise in pressure that becomes even steeper as smaller increments in fluid cause a disproportionate increase in pericardial pressure. (*Right*) a slower rate of pericardial filling takes longer to exceed the limit of pericardial stretch because there is more time for the pericardium to stretch and for compensatory mechanisms to become activated. Adapted from Spodick.³

However, inspiration reduces the pressure in the extra-pericardial pulmonary vessels, causing pulmonary pooling of blood and reducing pulmonary venous return to the

compressed left atrium and ventricle.^{6,9,17} This negatively impacts left-heart filling, stroke volume, and mean arterial pressure (fig. 5A).¹⁵ The respiration-induced hemodynamic

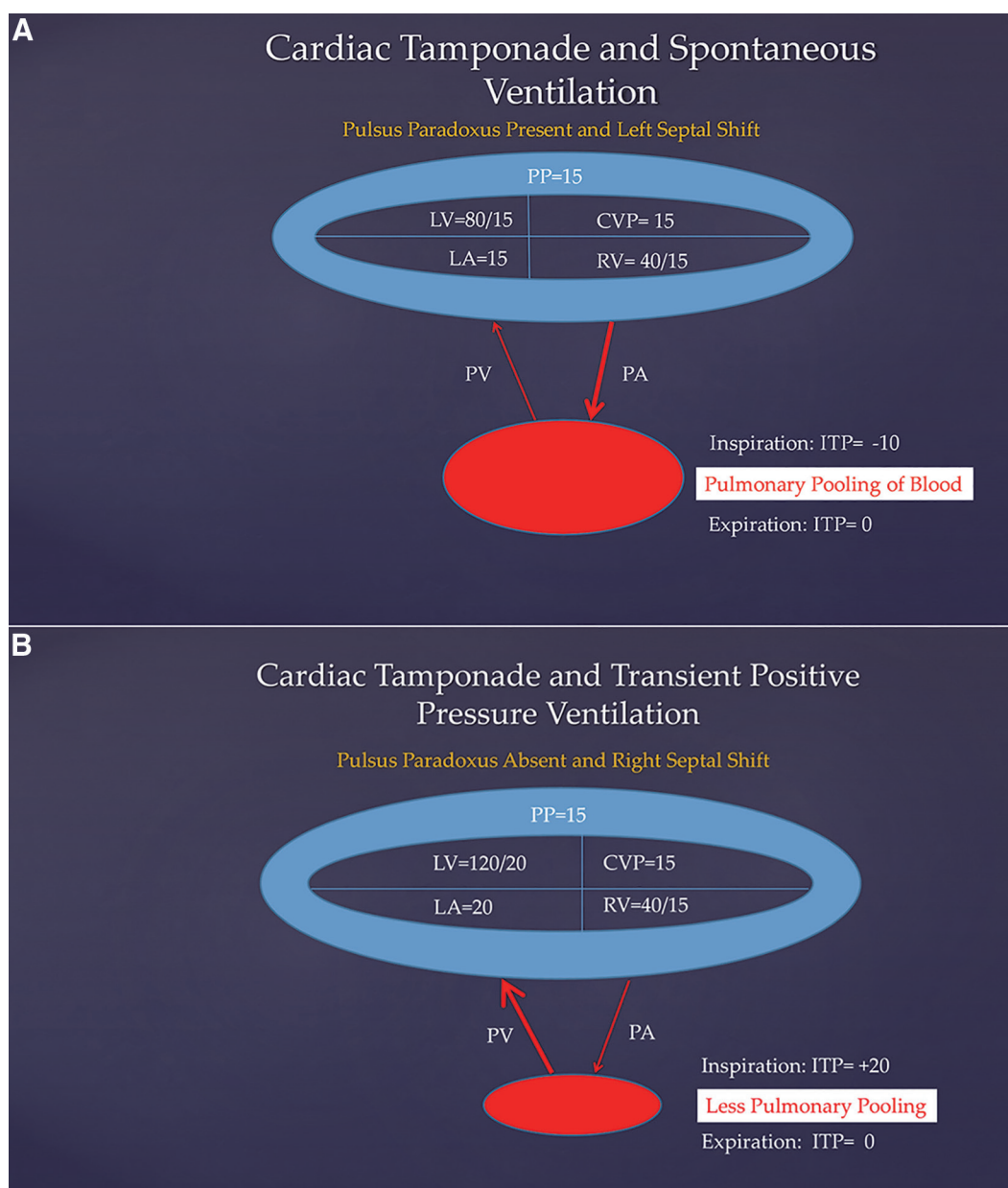


Fig. 5. Diagram showing global tamponade, illustrating pericardial pressure and differences in cardiac systolic and diastolic pressure, in pulmonary arterial and pulmonary venous blood flow, and pulmonary pooling of blood (A) during inspiration and spontaneous ventilation and (B) during inspiration and positive-pressure ventilation. All pressures are shown in millimeters mercury. Adapted from Royster *et al.*¹⁵ CVP, central venous pressure; ITP, intrathoracic pressure; LA, left atrium; LV, left ventricle; PA, pulmonary artery; PP, pericardial pressure; PV, pulmonary vein; RV, right ventricle.

changes during tamponade only apply to heartbeats during inspiration and expiration and are dependent on the heart rate-to-respiratory rate ratio as the blood flows in series through the pulmonary circulation.²¹ Eventually, as pericardial pressure further increases in phase III, all cardiac chambers are impacted, and diastolic pressures equilibrate (fig. 5A).¹⁵

Low-pressure Tamponade and Fluid Therapy

Animal studies have shown that cardiac tamponade can also develop with low intrapericardial pressure. In a hypovolemic patient, tamponade can develop when relatively low pericardial pressure exceeds right-heart pressure.^{22,23} These patients may not exhibit all clinical manifestations

of tamponade (tachycardia, jugular venous distention, and paradoxical pulse) leading to a misdiagnosis of clinical stability. Nonetheless, volume resuscitation to increase right-sided chamber pressure and pericardial drainage can result in marked hemodynamic improvement.²⁴ Low-pressure tamponade has been reported in patients with pericarditis, malignancy, and hypothyroidism and post dialysis and transcatheter aortic valve replacement.^{25–27} Patients with pericardial effusion often have symptoms of dehydration, have been treated with diuretics or recent dialysis, or have been treated with vasodilators, which reduce intravascular volume. Vasodilation induced by general anesthesia could theoretically precipitate tamponade physiology in a patient with pericardial effusion by reducing intracardiac pressure less than pericardial pressure.

Patients often have pericardial pressure less than 7 mmHg at cardiac catheterization, with echocardiographic findings of right-heart diastolic collapse but without classic clinical signs.^{28,29} Left-heart diastolic collapse has even been reported in low-pressure tamponade.³ However, inferior vena cava distension is often not present.²⁸

Anesthesiologists should be aware of this pathophysiology in hypovolemic patients, and treatment of low-pressure tamponade should consist of increasing intracardiac pressure with fluid therapy to counteract high pericardial pressure.³⁰ Fluid administration up to 500 ml appears to be appropriate, but greater amounts may not be effective in many patients.³¹ Fluid therapy may increase right-heart pressure as well as pericardial pressure in low-pressure tamponade and may not improve hemodynamics in all patients.³¹ A systolic blood pressure of less than 100 mmHg at baseline is predictive of a positive fluid response with a significant increase of mean arterial pressure of around 10% and cardiac index of as much as 20%.²⁹ Compared with high-pericardial-pressure tamponade, maintaining spontaneous ventilation may reduce pericardial pressure more and therefore have a greater effect on maintaining mean blood pressure.²⁵

Inotropes and Vasopressors

Drugs for hemodynamic support may be necessary in some patients with phase II and phase III pathophysiology prior to definitive treatment for cardiac tamponade. The basic principles involve maintaining an increased heart rate, improving cardiac output, and maintaining adequate systemic perfusion pressure. Although endogenous sympathetic output is usually maximized and tachycardia is common, some patients may be on chronic therapy to reduce heart rate (beta blockade), may have reduced left ventricular function, or may need general anesthesia and need inotropic and/or pressor support.

A study of isoproterenol in an animal model of tamponade found that isoproterenol increased heart rate and cardiac output and decreased right atrial and pericardial pressures while maintaining mean blood pressure.³² Reducing heart

size with an inotropic agent can theoretically reduce pericardial pressure. In another study in spontaneously breathing animals with tamponade, isoproterenol increased heart rate and cardiac output and reduced right atrial and intrapericardial pressure, and mean blood pressure.³³ The same study compared isoproterenol in eight patients with cardiac tamponade before pericardiocentesis with pulmonary artery catheters and found a small increase in cardiac output with isoproterenol.³³ Intracardiac pressure did not decrease. In another animal study, dobutamine increased cardiac index, oxygen delivery, and oxygen consumption with a lower blood lactate concentration as compared with norepinephrine.³⁴

Norepinephrine, in two animal studies of tamponade, resulted in variable effects with a consistent increase in mean blood pressure and an inconsistent increase in cardiac output at higher doses without reducing right atrial pressure or pericardial pressure.^{32,33} In humans, norepinephrine did not increase cardiac output but increased mean blood pressure. Dopamine tends to increase heart rate and cardiac output with little changes in other cardiac pressure in both animals and humans.^{32,33} Dopamine, norepinephrine, and isoproterenol all increased coronary blood flow, as measured by radioactive microspheres in animals.³³

Inotropic drugs with mild vasodilator effects appear to be advantageous in tamponade to increase cardiac output and reduce intracardiac pressure and possibly pericardial pressure. Isoproterenol, dobutamine, and low doses of epinephrine appear to have these preferred effects for treating tamponade. However, mean arterial pressure should be maintained as well to support cardiac and cerebral perfusion pressure. Adding a vasoconstrictor to an inotrope/vasodilator and titrating to keep mean arterial pressure greater than 65 mmHg appears to be advantageous hemodynamically.

Positive-pressure Ventilation and Anesthesia

Although spontaneous ventilation is often recommended for patients with cardiac tamponade, patients may develop significant hypoxemia and hypercarbia and require endotracheal intubation and positive-pressure ventilation.³⁵ Case reports indicate that intubated patients with positive-pressure ventilation required for pericardiocentesis or pericardial windows often do well hemodynamically.³⁶ Although spontaneous ventilation helps maintain systemic venous return and pulmonary blood flow, with pulmonary pooling of blood in tamponade, positive-pressure ventilation can further improve pulmonary venous return to the left ventricle, left ventricular stroke volume, and mean arterial pressure in animals (fig. 5B).^{15,37} Positive-pressure ventilation can actually decrease, eliminate, or reverse pulsus paradoxus in humans with tamponade.

A case series of pericardial drainage procedures reported by anesthesiologists comparing sedation, local anesthesia, and spontaneous ventilation with general anesthesia and positive-pressure ventilation found no difference in

outcomes.³⁸ However, general anesthesia patients required more vasopressor support, likely because of blunting of compensatory mechanisms impacting the pathophysiology.³⁸ One case report of acute tamponade in a sedated patient who was unresponsive to vasopressors demonstrated improved hemodynamics after general anesthesia, intubation, and positive-pressure ventilation.¹⁵ The authors theorized that improved pulmonary venous return and catecholamine delivery to the left heart with positive-pressure ventilation helped restore systemic blood pressure.¹⁵ Once the amount of blood that has pooled in the pulmonary circulation is reduced with positive-pressure ventilation, the heart may become more dependent on systemic venous return to the right heart to maintain cardiac volumes to both the right and left heart, and decreases in mean systemic pressure may occur.

General anesthesia, intubation, and positive-pressure ventilation may be required for respiratory management or surgical decompression (table 3). Patients with pericardial effusion in phase I (pericardial pressure less than right intracardiac pressure) should tolerate anesthesia well unless vasodilation and reduction in preload occur; tamponade physiology could then develop. Fluid therapy should be beneficial. Anesthesia providers should anticipate blunting of compensatory sympathetic tone in patients in phase II (pericardial pressure greater than right intracardiac pressure) and phase III (pericardial pressure greater than right and left intracardiac pressure) with general anesthesia. A preinduction fluid challenge in case intracardiac pressure is low to increase preload, inotropic and vasoactive drugs to maintain heart rate and systemic vascular resistance, and a preinduction arterial catheter to monitor for hypotension should be considered. Slow titration of anesthetic agents will avoid higher serum levels of drug, which will also help avoid loss of the sympathetic response to tamponade pathophysiology. Respiratory management should include decreased tidal volume and higher respiratory rate to maintain minute ventilation and to reduce the negative impact on systemic venous return.³⁹ Pressure-controlled ventilation may be

preferred. Positive end-expiratory pressure may help augment venous return to the left cardiac chambers, which is the primary cause of systemic hypotension in tamponade, but should be used cautiously because of the risk of reducing right-sided venous return.

Pericardial Decompression Syndrome

Pericardiocentesis or surgical pericardial drainage can result in hemodynamic instability and/or pulmonary edema post pericardial decompression.^{40,41} This syndrome develops in 50% of patients immediately or within 60 min of pericardial drainage but can occur up to 48 h later.⁴² The incidence is estimated to be less than 5% but with a mortality rate as high as 30% usually in patients undergoing surgical drainage.⁴³

After drainage, patients can present with left ventricular failure, biventricular failure, or right ventricular failure with noncardiogenic pulmonary edema.^{42,44} There is an increased risk for this syndrome in patients with large effusions that develop slowly, patients with rapid removal of large amounts of pericardial fluid, patients with reduced ventricular function, and patients with malignant effusions who receive radiation therapy.^{45,46} The incidence of this syndrome may be reduced, especially in patients with large effusions, if pericardial fluid is drained slowly until tamponade physiology is no longer present and hemodynamics return to normal.⁴³ A pericardial drain should be placed, with the remainder of the pericardial fluid removed over the next 24 to 48 h. Anesthesiologists should be prepared to diagnose and treat acute pulmonary edema post pericardial decompression in these patients.

The mechanisms involved in pericardial decompression syndrome are poorly understood (fig. 6).⁴⁷ The removal of a large amount of pericardial fluid can result in rapid venous return to the right ventricle and septal shift to the left, compromising left-sided filling and cardiac output. Additionally, increases in pulmonary venous return to the left ventricle with a high systemic vascular resistance could result in left ventricular failure and pulmonary edema, especially in patients with preexisting reduced left ventricular function.⁴⁰ With sudden increases in atrial and ventricular distension from increased venous return, an acute rise in ANP and BNP secretion can result in increased pulmonary capillary hydrostatic pressure and capillary permeability.^{48,49} Furthermore, myocardial reperfusion injury can occur when coronary blood flow returns to ischemic myocardium that resulted from reduced coronary flow secondary to high pericardial pressure during tamponade.^{50,51} Finally, some have postulated a potentially adverse response to the normal catecholamine surge during tamponade. The normal response to hypotension and reduced cardiac output associated with tamponade is a marked increase in sympathetic tone resulting in tachycardia and release of endogenous catecholamines. This sympathetic response may develop slowly over time as the pericardial effusion increases in size and develops tamponade physiology.

Table 3. Considerations for Patients Requiring General Anesthesia for Pericardial Surgery During Cardiac Tamponade

- Fluid challenge to stabilize hemodynamics if intracardiac pressure is reduced in low-pressure tamponade.
- Inotrope/vasopressors to maintain heart rate, contractility, and systemic resistance and other compensatory physiology. Intracardiac and pericardial pressures may decrease.
- Preinduction arterial catheter.
- Surgical site prepped and draped for urgent pericardial window.
- Slow titration of anesthetic induction agents to avoid loss of compensatory physiology.
- Intubation and ventilation with low peak pressure; Consider pressure control ventilation.
- Use positive end-expiratory pressure cautiously.
- Transesophageal echocardiography to assess pericardial drainage.

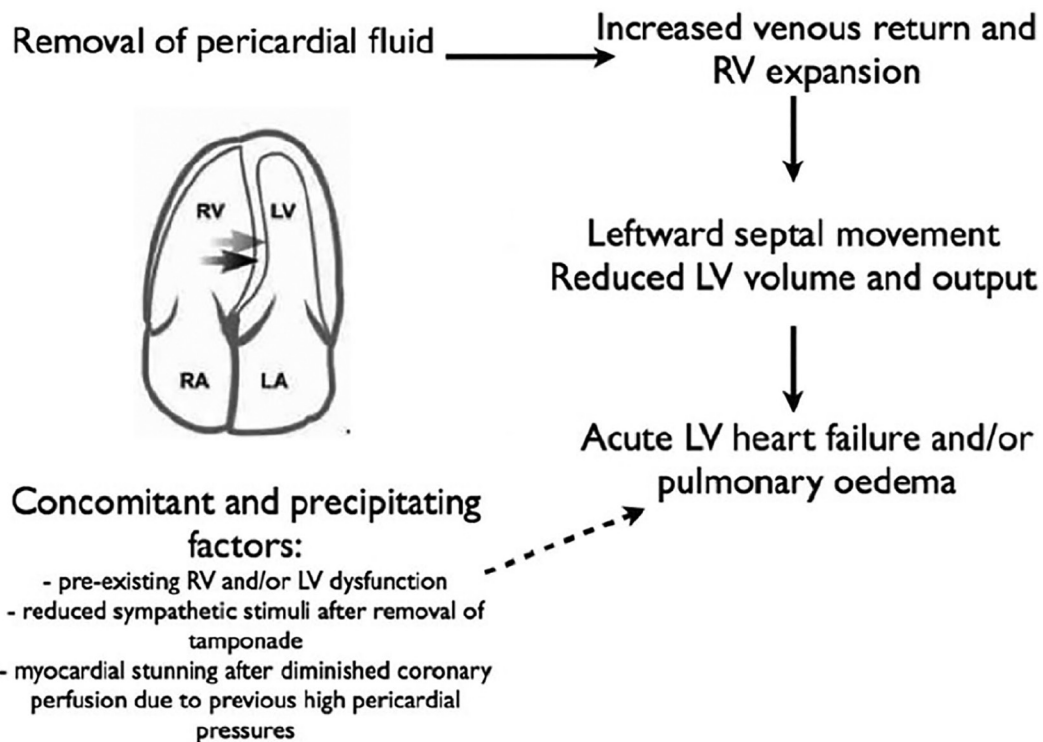


Fig. 6. Pericardial decompression syndrome. Intracardiac pressure increases, and atrial and ventricular dilation with release of atrial natriuretic peptide and brain natriuretic peptide may also play a role in the generation of pulmonary edema post decompression.⁴³ LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Stress-related cardiomyopathy could develop, which then manifests as acute right or left heart failure when the pericardium is drained and venous return increases.^{52,53}

Chest Compressions and CPR

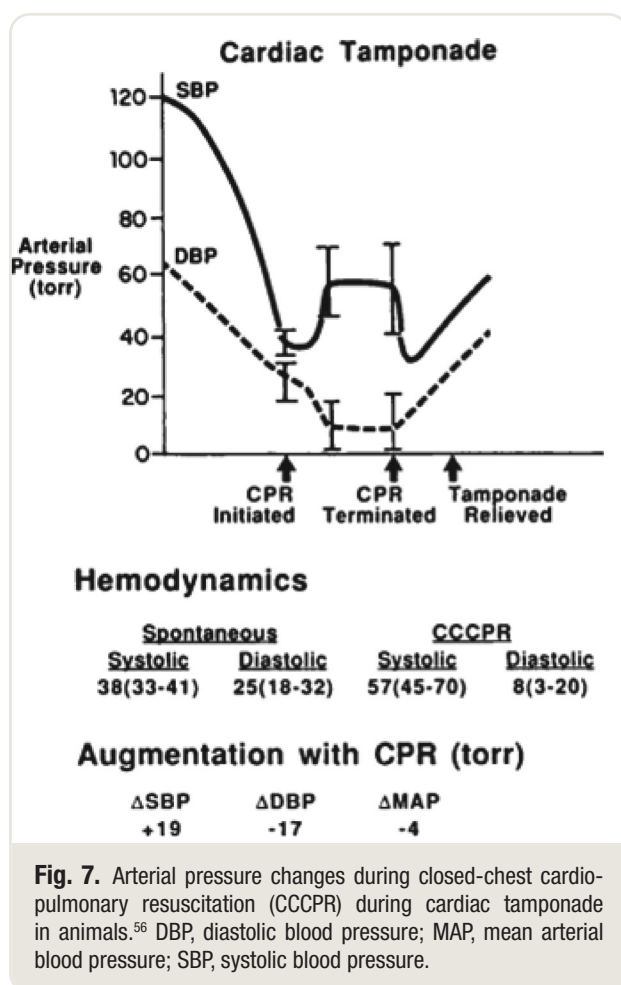
The physiology of chest compressions during CPR involves either the cardiac compression theory, where the heart is compressed and pumps blood into the aorta and pulmonary arteries, or the thoracic pump theory, where the intrathoracic pressure increases and compresses all thoracic vessels and moves blood out of the thorax into the systemic arterial circulation.⁵⁴ However, both of these theories may be effective depending on the patient. The pathophysiology of cardiac tamponade, as illustrated by electrical alternans on the electrocardiogram or the heart moving with each beat within a large pericardial effusion during an echocardiogram, suggests that it would be more difficult to compress the heart.⁵⁵ A study of cardiac tamponade and CPR in baboons demonstrated that chest compressions resulted in an increase in pericardial pressure, a clinically insignificant increase in systolic pressure, and a decrease in diastolic pressure and mean arterial pressure and was thought to be ineffective (fig. 7).⁵⁶

However, in clinical practice, several case reports have documented successful chest compressions during CPR in humans, suggesting the thoracic pump theory to be

effective.^{54,55} Additionally, pericardial tears have been reported that created a communication with the left pleural space and resulted in chest compressions relieving tamponade.^{57–59} Perforation of the pericardium by fractured rib fragments can also reduce pericardial effusion.⁵⁷ New left-sided pleural effusion was detected in almost all case reports of pericardial tears.^{57–59} However, other case reports have described chest compressions causing injury to the pericardium, pericardial effusion, and tamponade.^{58–60} Injuries are more likely with aggressive compressions, which result in depression of the sternum greater than 6 cm.⁶⁰ In a patient requiring CPR for resuscitation due to tamponade, anesthesia providers should move urgently to definitive treatment with pericardiocentesis or a surgical pericardial window.

Conclusions

Cardiac tamponade is a respirophasic condition that results in a pathophysiology that causes pericardial compression of the cardiac chambers, which can lead to profound hemodynamic compromise. The hallmark physical finding of an exaggerated change in systolic blood pressure (pulsus paradoxus) driven by inspiratory reduction in left ventricular filling distinguishes this dangerous condition from other heart failure states. Interestingly, patients are often dyspneic due to pulmonary venous pooling of blood, but the absence of significant



chamber stretch (due to pericardial compression) limits natriuretic peptide secretion, which may decrease the presence of pulmonary edema. Echocardiography is crucial for diagnosis and shows significant respiratory variation in mitral and tricuspid inflow velocity and diastolic chamber collapse suggestive of hemodynamically significant increases in pericardial pressure.

The clinical management of tamponade hinges on pericardial decompression, although volume resuscitation can temporize hemodynamic instability, especially in cases of low-pericardial-pressure tamponade. General anesthesia and intubation may reduce sympathetic tone and other compensatory mechanisms and result in hemodynamic instability, which may require fluid and inotropic/vasopressor support. However, there are potential transient benefits of positive-pressure ventilation by increasing left ventricular filling from pulmonary pooling of blood, which can improve the systemic circulation of vasopressors and systemic blood pressure and even eliminate the paradoxical pulse in some patients. Pericardial decompression can dramatically improve hemodynamics, but anesthesiologists should be aware of the risk of pericardial decompression syndrome, a constellation of hemodynamic instability and

progressive pulmonary edema, which can occur if pericardial drainage is too robust or rapid, usually occurring soon after surgical decompression. Chest compressions may not be as effective in tamponade, but there are numerous case reports of successful chest compressions during CPR and even tamponade relief with chest compressions, and they should be considered by anesthesiologists, if necessary, prior to emergency decompression.

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